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Wheat and Rye Stem Rust

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I. Introduction

Puccinia graminis Pers. is the cause of several rusts of important cereal crops. Although in antiquity *P. graminis* may have been a single population, the cultivation of groups of dissimilar crops (wheat, oats, barley, and rye) resulted in the development and increase of different pathogen genotypes capable of attacking these crops. One may assume specialization occurred through selection of particular crops. Although crossing between cultures adapted to the same host could result in improved adaptation in the progeny, crossing between *formae speciales* would result in many progeny avirulent on both crops because of the recessive nature of virulence. Thus, the *formae speciales* have developed and been maintained as nearly separate populations of *P. graminis*.

A genetic relationship still exists, however, between many of the *formae specialis*. A diallel series of crosses was made by Johnson (1949) between pairs of f. sp. *tritici, secalis, avenae, agrostidis,* and *poae.* Crosses between f. sp. *tritici* and *secalis* were relatively fertile. Progeny from such crosses often are virulent on a limited number of host genotypes of both rye and wheat. Crosses within cultures of either f. sp. *tritici* or *secalis* can occasionally result in an F_1 culture that can be classified in the other *forma specialis*.

A. THE DISEASES

1. *Wheat Stem Rust*

Stem rust of wheat is caused by *Puccinia graminis* Pers. f. sp. *tritici.* This disease is also known as black stem rust or summer rust. Early references to stem rust or a disease now thought to be stem rust were reviewed by Chester (1946). Recently, urediospores were found in Israel that have been dated as 3300 years old (Kislev, 1982).

The damage caused by wheat stem rust can be more spectacular than any other cereal disease. Millions of hectares of a seemingly healthy crop with a high yield potential can be totally destroyed in less than a month. These types of epidemics on a smaller scale have been observed in nearly every country where wheat is grown. However, it was the continental epidemics that occurred in Australia and North America that showed the potential destructiveness of wheat stem rust. Stem rust of wheat is probably the most thoroughly studied plant disease. Certainly, more is known of the genetics of host resistance, frequency

and distribution of pathogen virulence phenotypes, disease epidemiology and the physiology, biochemistry, and histology of the host–pathogen interaction than for any other plant disease. Although this chapter does not cover all that is known about stem rust of wheat, this and other chapters in this two-volume treatise will review much of the available information.

2. *Rye Stem Rust*

Stem rust of rye caused by *P. graminis* f. sp. *secalis* has been a minor disease worldwide. However, in Brazil in 1982, it totally destroyed the scattered fields of rye throughout the southern half of the country. Stem rust of rye has caused little damage in the United States (Roelfs, 1978), and historically, it probably has been the most serious as a disease in northern Europe. The main reasons generally given for its minor importance worldwide are the limited areas of rye grown in comparison with other cereals and the genotypic variation in rye as a cross-pollinated crop. However, in Brazil where only a few isolated rye fields existed, a severe epidemic was not averted. Rye stem rust was recently introduced into Brazil, and the ryes are old land cultivars. Stem rust has also become more severe on rye in Australia (Tan *et al.*, 1975). Perhaps the rye cultivars have not accumulated or maintained factors for resistance, or perhaps the introduced pathogen is particularly virulent or aggressive. Kingsolver *et al.* (1959) were able to initiate severe epidemics of stem rust on rye in the eastern United States by artificial inoculation. Most of the interest in *P. graminis* f. sp. *secalis* has been due to its close relationship to f. sp. *tritici* and to their putative hybrids (see Chapter 10, this volume) and its evolutionary significance (Green, 1971). However, the transfer of rye chromosomes sections to wheat and the possibility of triticale becoming a crop has also renewed our interest in rye stem rust.

Stakman *et al.* (1930) found that when wheat stem rust race 36 was crossed with rye stem rust race 11, a large range of pathogenic types was obtained including some common races of wheat stem rust. Johnson (1949) crossed races 1 and 30 of wheat stem rust with rye stem rust and recovered race 111 of wheat stem rust among others. In Australia, Watson and Luig (1962) selfed rye stem rust and obtained cultures virulent on Little Club (*SrLC*), Eureka (*Sr6*), and Yalta (*Sr11*). Parasexual recombinants also occur between rye and wheat stem rust (Watson and Luig, 1959). Thus, members of these two *formae speciales* seem to have many genes in common.

3. *Barley Stem Rust*

Stem rust of barley is caused by *P. graminis* f. sp. *tritici* and in North America also by f. sp. *secalis*. Epidemics of barley stem rust were rare historically except when wheat was severely rusted (Roelfs, 1978). Seedlings of most, if not all, barley cultivars have a moderate level of resistance at 18°C (Steffenson *et al.*, 1982b). This resistance is expressed in the seedling stage by a mesothetic response. In adult plants, the resistance is expressed by a reduction in lesion size and number (Steffenson, 1983). This level of resistance in barley, when accompanied by its relatively early maturity and ability to grow at low temperatures, generally provides protection from endogenous inoculum of f. sp. *tritici*.

A single gene with a major effect and one or more genes with lesser effects have been identified in barley. The gene with a major effect was designated as the *T* gene because it provided resistance to *forma specialis tritici* (Powers and Hines, 1933). The *T* gene occurs in the cultivars Chevron and Peatland, selections from a bulk seed lot from the Swiss Seed Experiment Station. Another source of this resistance is the cultivar Kinred, which was selected as an offtype plant from a field of Wisconsin Pedigree 37. The *T* gene resistance has remained effective, even though widely used in the northern United States since Peatland was released in 1926. The *T* gene resistance against some races of *P. graminis* f. sp. *tritici* is less effective in some cultivars than others (Steffenson *et al.*, 1982a,b).

As the frequency of wheat stem rust has decreased in North America, the proportion of isolates of *P. graminis* f. sp. *secalis* isolated from barley has increased (Green, 1971). The level of resistance in barley against rye stem rust seems to be less than against wheat stem rust in North America (Green, 1971; Steffenson, 1983). The *T* gene offers no protection against rye stem rust, and most cultivars are damaged in inoculated nurseries (Steffenson *et al.*, 1982a). Currently, inadequate inoculum exists of *P. graminis* f. sp. *secalis* to threaten North American barley production. However, because this *forma specialis* is virulent on many species of native and introduced grasses, inoculum levels may change. Resistance to *P. graminis* f. sp. *secalis* exists in the cultivars Black Hulless, Heitpas-5, and Valkie (Steffenson *et al.*, 1982a).

In Australia, putative hybrids between *P. graminis* f. sp. *tritici* and f. sp. *secalis* seem to have specialized on barley (Chapter 10, this volume).

4. Puccinia graminis *on the Alternate Hosts*

Many species of *Berberis*, *Mahonia*, and their hybrid (× *Mahoberberis*) are susceptible to *Puccinia graminis* (Table I). Little specialization occurs on the alternate hosts but is known to occur (Waterhouse, 1929b; Green and Johnson, 1958; Johnson and Green, 1954). Even on susceptible bushes only the tissue (2 weeks old or less) is normally susceptible (Melander and Craigie, 1927). The most important of the susceptible species has been *Berberis vulgaris* L., although other susceptible species exist throughout most of the world. The resistant species of *Berberis* (Table II) tend to have a thick cuticle (Melander and Craigie, 1927), although in a few *Berberis* species and some *Mahonia* species attempts to infect the bush resulted in small hypersensitive flecks, which is an indication of a physiological mechanism of resistance.

Table I

Species of *Berberis, Mahonia,* and × *Mahoberberis* Susceptible to *Puccinia graminis*[a]

Berberis	*Berberis*
acuminata Franch.	*crataegina* DC.
aemulans Schneid.	*cretica* L.
aetnensis Presl.	× *declinata* Schrad.
aggregata Schneid.	× *declinata* var. *oxyphylla* Schneid.
× *alksuthiensis* Ahrendt	*delavayi* Schneid.
amurensis Rupr.	*diaphana* Maxim.
angulosa Wall. ex Hook f. et Thoms.	*dictyoneura* Schneid.
aristata DC.	*dictyophylla* Franch.
asiatica Roxb. ex DC.	*dielsiana* Fedde
atrocarpa Schneid.	× *durobrivensis* Schneid.
× *barbarossa* Watson ex Ahrendt	*edgeworthiana* Schneid.
bergmanniae Schneid.	× *emarginata* Willdenow
boschanii Schneid.	× *emarginata* var. britzensis Schneid.
brachypoda Maxim.	*empetrifolia* Lam.
bretschneideri Rehd.	*fendleri* Gray
× *cerasina* Schrad.	*francisci-ferdinandi* Schneid.
chinensis Poir.	*glaucocarpa* Stapf
chitria Lindl.	*globosa* Benth.
chrysosphaera Mulligan	*henryana* Schneid.
consimilis Schneid.	*heteropoda* Schrenk
coriaria Royle ex Lindl. var. *patula* Ahrendt	*holstii* Engler
	hookeri Lemaire

(continued)

Table I (*Continued*)

Berberis	*Berberis*
hookeri var. *viridis* Schneid.	*soulieana* Schneid
humido-umbrosa Ahrendt	× *spaethii* Schneid.
hypokeriana Airy-Shaw	*stiebritziana* Schneid.
ilicifolia Forst.	*suberecta* Ahrendt
jamesiana Forrest et W.W. Sm.	*thibetica* Schneid.
× *laxiflora* Schrad.	*turcomannica* Kar. ex Ledeb.
× *laxiflora* var. langeana Schneid.	*umbellata* Wall. ex G. Don
lecomtei Schneid.	× *vanfleetii* Schneid.
lycium Royle	*vernae* Schneid.
× *macracantha* Schrad.	*vulgaris* L.
× *meehanii* Schneid. ex Rehd.	*wilsonae* Hemsl.
mitifolia Stapf	*wilsonae* var. *parvifolia* (Sprague) Ahrendt
morrisonensis Hayata	*wilsonae* var. *stapfiana* (Schneid.) Schneid.
× *notabilis* Schneid.	*yunnanensis* Franch.
nummularia Bunge	*Mahonia*
oblonga (Regel) Schneid.	*fremontii* (Torr.) Fedde
orthobotrys Bienert ex Aitch.	*haematocarpa* (Wooton) Fedde
× *ottawensis* Schneid.	*napaulensis* DC.
poiretii Schneid.	*nevinii* (Gray) Fedde
polyantha Hemsl.	*swaseyi* (Buckley) Fedde
prattii Schneid.	*trifoliolata* (Moric.) Fedde
pruinosa Franch.	× *Mahoberberis*
× *rubrostilla* Chittenden	*neubertii* (Baum.) Schneid.
× *serrata* Koehne	
sibirica Pall.	
sieboldi Miq.	

[a]From Ahrendt (1961).

B. EPIDEMIOLOGY

Details on epidemiology in Australia–New Zealand, Europe, the Indian subcontinent, and North America are covered in separate chapters in this volume. The earlier reports of Chester *et al.* (1951), Rajaram and Campos (1974), and Hogg *et al.* (1969) contain additional historical information on these areas. In the rest of Asia, South America, and Africa, continental studies are not available.

II. Life Cycle

The life cycle of *P. graminis* has been widely studied, but unanswered questions still remain. The first drawing of the fungus was

Table II

Species of *Berberis, Mahonia,* and × *Mahoberberis* Resistant to *Puccinia graminis*[a]

Berberis	*Berberis*
arido-calida Ahrendt	*sargentiana* Schneid.
beaniana Schneid.	*sikkimensis* (Schneid.) Ahrendt
buxifolia Lam.	× *stenophylla* Lindl. var. *diversifolia* Ahrendt
buxifolia var. *nana* A. Usteri	× *stenophylla* var. *gracilis* Ahrendt
calliantha Mulligan	× *irwinii* Byhouwer
candidula Schneid.	*taliensis* Schneid.
cavaleriei Léveillé	*temolaica* var. *artisepala* Ahrendt
× *chenaultii* (Hort. ex Cat. L. Chenault) Ahrendt	*thunbergi* DC.
	thunbergi var. *agenteo-marginata* Schneid.
circumserrata Schneid.	*thunbergi* var. *atropurpurea* Chenault
concinna Hook. f. et Thoms.	*thunbergi* var. *erecta* (Rehd.) Ahrendt
coxii Schneid.	*thunbergi* var. *maximowiczii* (Regal) Regal
darwinii Hook.	*thunbergi* var. *minor* Rehd.
dasystachya Maxim.	*thunbergi* var. *pluriflora* Koehne
dubia Schneid.	*triacanthophora* Fedde
franchetiana Schneid.	*verruculosa* Hemsl. et Wils.
gagnepainii Schneid.	*virgetorum* Schneid.
gilgiana Fedde	*xanthoxylon* Hasskarl ex Schneid.
gyalaica Ahrendt	*Mahonia*
heterophylla Juss. ex Poir.	*amplectans* Eastwood
× *hybrido-gagnepainii* Suringar	*aquifolium* (Pursh) Nutt.
insignis Hook. f. et Thoms.	*bealei* (Fort.) Carr.
julianae Schneid.	*dictyota* (Jepson) Fedde
kawakamii Hayata var. *formosana* Ahrendt	*fortunei* (Lindl.) Fedde
	japonica (Thunb.) DC.
koreana Palib.	*lomariifolia* Takeda
lempergiana Ahrendt	*nervosa* (Pursh) Nutt.
lepidifolia Ahrendt	*pinnata* (Lag.) Fedde
linearifolia Phil.	*piperiana* Abrams
× *lologensis* Sandwith	*pumila* (Greene) Fedde
manipurana Ahrendt	*repens* G. Don
pallens Franch.	× *Mahoberberis*
potaninii Maxim.	*aquicandidula* Krüssmann
replicata W. W. Sm.	*aquisargentiae* Krüssmann
sanguinea Franch.	*miethkeana* Melander et Eade

[a]From Ahrendt (1961).

made by Fontana (1932). DeBary (1866) showed that the two fungi, *P. graminis* on cereals and *Aecidium berberidis* on barberry, were different stages of the same organism. A simple life cycle of *P. graminis* is shown in Fig. 1. Readers interested in more structural detail should consult the appropriate chapters in Volume I of this treatise or Lit-

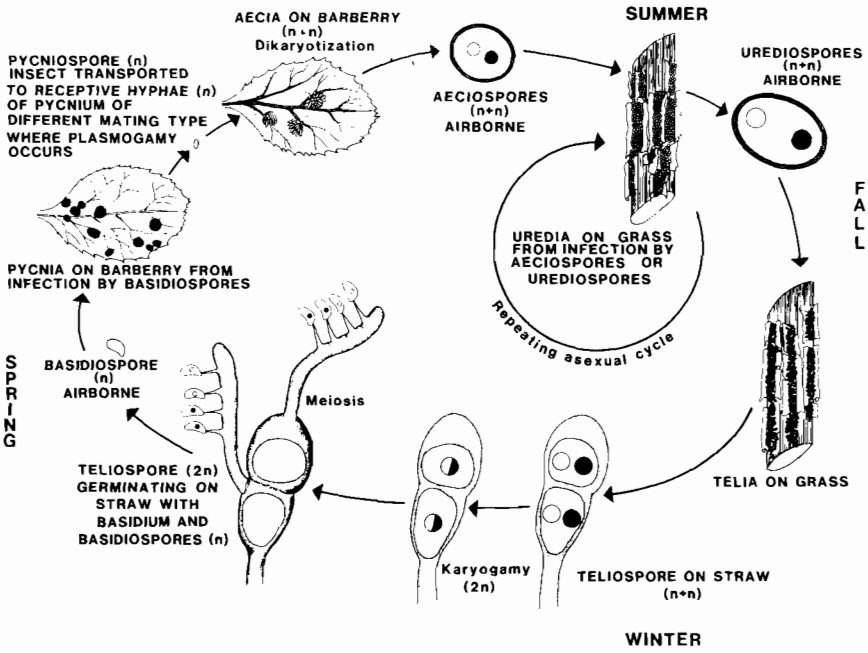

Fig. 1. A simplified life cycle of *Puccinia graminis*. The nuclear condition is shown by n (haploid), n + n (dikaryotic), and 2n (diploid). Spores are all drawn to approximately the same scale. For details and variations in structures and developmental events, see Volume I, Chapters 11 and 12. Germinating teliospore adapted from Buller (1924).

tlefield and Heath (1979) and Littlefield (1981). The life cycle description in this chapter emphasizes the effects of the life cycle on disease development.

A. BASIDIOSPORES

Basidiospores are formed on sterigma on each cell of the basidium. The spores are actively discharged a few centimeters from the sterigma at maturity (Buller, 1958). Basidiospores are small, 7.6 × 6 μm (D. L. Long, personal communication), hyaline, and oval-shaped. They can develop and are released within 20–25 min after the teliospore is moistened (Craigie, 1940); however, some viable teliospores may not germinate for 3–6 or more days (Cotter, 1932). Alternate wetting and drying, freezing and thawing may help to induce teliospore germination. The basidiospores are wind-borne to the alternate host, species of

susceptible *Berberis* and *Mahonia*, but seldom cause infection more than 180 to 270 m from the source. Basidiospores are short-lived, surviving only a few hours under ideal conditions. Therefore, basidiospores germinate rapidly by producing a germ tube with a terminal appressorium from which an infection peg develops and directly penetrates the epidermal cell wall. Most of the infections occur on the upper leaf surface, although infections on the berries, stems, and other plant surfaces do occur. Leaves of susceptible cultivars often appear to become resistant to penetration about $2\frac{1}{2}$ weeks after the leaf bud unfolds.

B. PYCNIOSPORES

Approximately 5 days after infection of the barberry by the basidiospore, the initial signs of pycnia development become visible (Cotter, 1932). At 7–14 days after infection, the small, 1.6×3.6 μm (D. L. Long, personal communication), haploid, unicellular pycniospores appear in a viscous fluid that forms at the pycnial opening. The pycniospore functions as a gamete and fuses with the receptive hypha that functions as the other gamete. To accomplish fertilization, pycniospores must be transferred to receptive hyphae of a pycnia of a different mating type. This transfer of pycniospores is by insects presumably attracted to the fluid in which the spores are suspended. The function of the pycniospores as sexual gametes was unknown until the work of Craigie (1927).

C. AECIOSPORES

Following fertilization, the nucleus of the pycniospore migrates through the monokaryotic hyphae until it reaches the protoaecium. This process requires 20–25 hr. Following dikaryotization, an aecium develops on the lower surface of the barberry leaf within 7 to 10 days after fertilization of the receptive hyphae (Cotter, 1932). The aeciospores represent the recombination products of the genetic process. The variation in virulence among aeciospores was first shown by Waterhouse (1929a). Aeciospores from an individual aecial horn generally have the same genotype, whereas aeciospores from another horn in the same aecium frequently have different genotypes. Aeciospores are dikaryotic, cylindrical, $16–23 \times 15–19$ μm, and are generally wind-borne over long distances. Aeciospores are forcibly released following the drying of previously wetted aecia. The optimum temperature for ger-

mination is 22°C (Novotelnova, 1935). Large numbers of spores are produced. Stakman (1923) estimated that an average-sized *Berberis vulgaris* bush in southern Minnesota had approximately 35,000 leaves, of which about 28,000 would be infected. On a single multi-infected leaf, 2.3 to 8 million aeciospores were produced. Thus, an average bush could be the source of 64×10^9 spores. Aeciospores have been found at altitudes of 2 km (Stakman and Harrar, 1957). Although the data are incomplete, initial infection from aeciospores is very heavy within a few meters of the source. Isolated infections can occur as far away as 100 m, and there is no reason to suspect that aeciospores are not transported as far as urediospores are (hundreds of kilometers). Aeciospores germinate on the gramineous host upon contact with free water. Germ-tube formation is followed by formation of an appressorium over a host stoma, and a penetration peg enters through the stomatal opening.

D. UREDIOSPORES

A uredium is produced after the successful infection of a grass host by *P. graminis*. Initial signs of the infection usually become visible 5–6 days after inoculation as a light colored spot. Sporulation usually follows infection by 7–14 days, with the shortest period at 30°C. The size of uredia and time required for their development are determined by host resistance–pathogen virulence, pathogen aggressiveness, host maturity, and infection density and environment, particularly temperature and light. Urediospores are dikaryotic, oblong, and about 26–40 × 16–32 μm in size. They can be transported long distances by the wind (Hirst and Hurst, 1967) in a viable condition (Luig, 1977). The terminal velocity (rate at which an object falls) of urediospores in still air ranges from 0.94 to 1.25 cm/sec, increasing with decreasing relative humidity and temperature (Weinhold, 1955; Ukkelberg, 1933). The spores may settle from the air under the influence of gravity, by impaction on objects when striking them, but are most effectively removed from the air by the scrubbing action of raindrops (Rowell and Romig, 1966).

Large numbers of spores are produced over a period of several weeks. A mature uredium produces daily about 23 μg (Katsuya and Green, 1967; Mont, 1970) of urediospores; 1 μg contains about 4.5×10^2 urediospores (Rowell and Olien, 1957). Thus about 10,000 urediospores per day are produced, and with a 5% disease severity (50 pustules per tiller), 5 kg of spores can be produced per hectare (Rowell and Roelfs,

1971). Most of the urediospores produced are deposited within the crop canopy and a large proportion of the remainder within 100 m of the source (Roelfs, 1972). However, due to the large numbers produced, significant numbers of urediospores reach heights of up to 3000 m and have been transported in a viable condition over great distances. The spores are relatively resistant to damage from temperatures 0°–40°C and can withstand greater extremes for short periods. Spores will remain viable at room temperatures for several weeks. Longevity of urediospores rapidly decreases with exposure to high relative humidities (over 80%), and longevity is prolonged at relative humidities of 20–30%.

The optimum temperature for urediospore germination and infection is about 18°C. An appressorium forms over a stoma within 3–6 hr. This part of the process can occur in the dark; however, for further development, approximately 10,000 lux of light is required over about 3 hr for the penetration peg to form and the appressorium to empty. During the light phase, for optimum development, temperatures should gradually rise from 18° to 30°C and a gradual drying should occur. Sporulating uredia usually are present in 10–14 days. Latent periods of 14–20 days are common in the field during early spring and have been observed to be as long as 30 days. The uredial cycle repeats itself every 14–21 days under normal field conditions. Because of varying environmental conditions in the field, some new infections appear almost daily; thus generations overlap and are generally indistinguishable.

E. TELIOSPORES

As host plants mature, uredia gradually develop into telia, producing teliospores. Teliospores are blackish-brown, oblong, diploid, two-celled spores about 40–60 × 16–23 μm. The spores remain attached to the telium by the stalk, and are rather resistant to environmental extremes (Cotter, 1932). Teliospores serve as the overwintering stage of the fungus in climates where freezes are common. Viability of teliospores is reduced with high temperatures, especially if this is accompanied by dry conditions. Teliospore viability varies with the host and pathogen genotypes involved.

Transport of teliospores is generally by man or water when infected wheat or rye straw is moved. The teliospore germinates after a period of weathering, alternating freezing and thawing and drying and wetting. However, under laboratory conditions, teliospores germinate ir-

regularly. Teliospores germinate by producing a basidium, during which time meiosis occurs and the haploid nuclei migrate into the basidium. A basidiospore develops on a sterigma, and a haploid nucleus migrates into the spore.

III. Disease Cycle

The available evidence points to separate populations of *P. graminis* f. sp. *tritici* in the United States, one that undergoes sexual reproduction annually and another that reproduces asexually (Roelfs and Groth, 1980). When the two populations separated is unknown. This may be similar to the development of two forms of *P. graminis* in eastern Europe at a subspecies level (Savile and Urban, 1982). Parasexual mechanisms have been proposed for *P. graminis;* however, if these processes occur in nature, most of the progeny must fail to compete satisfactorily with the preexisting population. The literature on these mechanisms was reviewed by Watson and Luig (1962). An exception may be the *P. graminis* f. sp. *tritici* and *P. graminis* f. sp. *secalis* hybrids (Luig and Watson, 1972) in Australia. In most areas of the world where stem rust is a disease of major importance, the pathogen survives without the sexual cycle. See Chapters 9–12, this volume, for Australia–New Zealand, Europe, the Indian subcontinent, and North America, respectively. Although published information is limited, the sexual cycle is not known to be important in the major wheat- and rye-producing areas of South America, Africa, or eastern or northern Asia.

A. ASEXUAL CYCLE

In mild climates the pathogen can reproduce by means of urediospores, and survives noncrop season(s) on volunteer cereal plants or on other gramineous hosts (Fig. 2). Survival is generally difficult for the pathogen during the noncereal growing season, and often the population is reduced to near or below the threshold of detection. However, a few surviving local uredia can produce local inoculum and thereby cause more infections than a severely rusted field 100 km distant from which spores must be transported. In mild climates the asexually reproducing pathogen generally survives the winter on the wheat or rye crop. In tropical climates it often exists as sporulating mycelium in the leaves, whereas in lower latitudes of temperate climates it may be

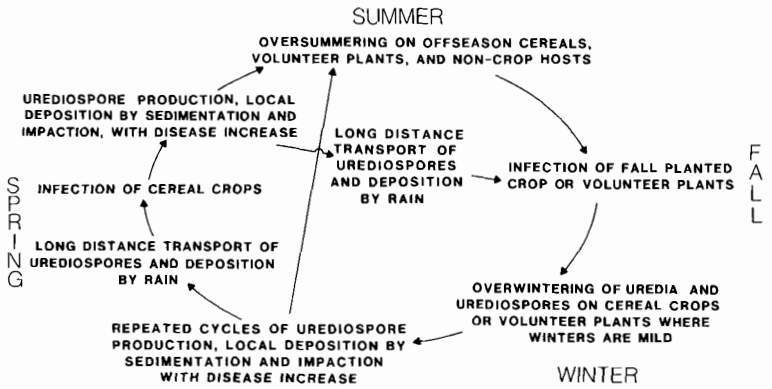

Fig. 2. The asexual disease cycle for stem rust, which is the most common source of disease worldwide. In areas with severe winters, the pathogen may not survive the winter and thus must be reintroduced by long-distance transport of urediospores from areas where winters are milder. Likewise, the pathogen may not survive the summers where it is very hot or dry, and thus must be reintroduced by long-distance transport of urediospores from areas where the summers are milder.

present as nonsporulating mycelium. In the central latitudes of the temperate zone, the pathogen generally fails to survive because the host tissue is winter killed. In these areas epidemics are generated from airborne inoculum from milder climates.

Epidemics seem to be relatively few in areas where stem rust overwinters on the crop itself; the crop is harvested in late spring before conditions are generally favorable for stem rust. Major epidemics are most frequent and severe on spring-planted cereals that grow during the spring and summer, and on fall-planted wheats that mature in the summer.

The major source of variation in the asexual population is by mutation, which is expressed as single gene changes. Although the number of mutations may be great because of the large number of urediospores produced, most mutations must be eliminated through selection (Roelfs and Groth, 1980). Other sources of variation are from exogenous inoculum (Luig, 1977) and parasexualism (Luig and Watson, 1972).

The asexual population is characterized by one or a few predominant phenotypes (Groth and Roelfs, 1982). Related phenotypes often exist (Roelfs and Groth, 1980) that reflect their origin through mutation. This relationship can be seen over the years as a gradual evolution with each new phenotype, clearly related to a previously existing one (Green, 1975) (Chapter 10, this volume).

In North America many of the asexual population phenotypes seem to be losing their ability to produce teliospores, and the teliospores often have a low viability (Roelfs, 1982). These factors could be among the reasons for the separation of the populations into distinct phenotype groups.

B. SEXUAL CYCLE

Where the sexual cycle functions, the pathogen overwinters as teliospores. If straw bearing teliospores is located near a susceptible alternate host (normally *B. vulgaris*), infection will normally occur. Teliospores usually germinate over a period of several days. Infections normally occur on the expanding barberry leaves that are produced in the spring (Fig. 3). The sexual cycle can furnish large amounts of inoculum in the form of aeciospores which initiate local epidemics, and then the resulting production of urediospores can cause regional epidemics (Roelfs, 1982).

The teliospores germinate better when they are produced under cool temperatures (<18°C) than under high temperature. Thus, the sexual populations normally have existed at cooler latitudes—that is, the northern United States, Canada, northern Europe, and at higher elevations farther south. Teliospores also occur on volunteer crop plants or other gramineous hosts late in the fall when temperatures are cool.

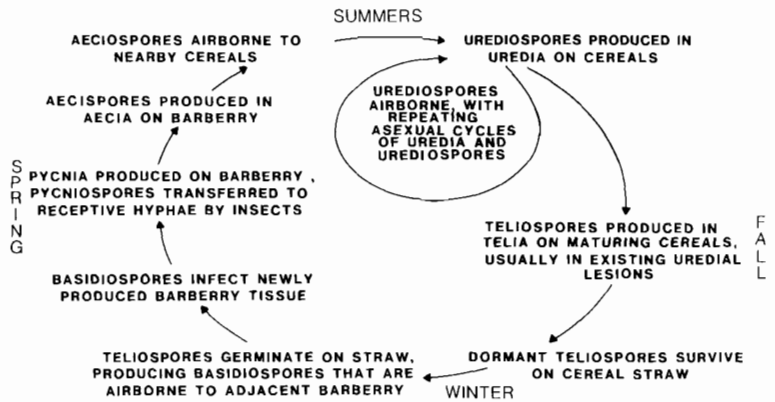

Fig. 3. The complete disease cycle (sexual and asexual) for stem rust, which historically occurred in eastern and northern Europe and in North America where barberry (the alternate host) was present. Viable teliospores are produced in regions without extreme heat as the cereal crop matures. Winters must be severe enough to break teliospore dormancy. For details of the life cycle, see Fig. 1; for details of the asexual disease cycle, see Fig. 2.

The sexually reproducing population is characterized by diversity. The total number of phenotypes is great (usually one phenotype per five or fewer isolates) (Roelfs and Groth, 1980; Groth and Roelfs, 1982). The various phenotypes in the populations tend to occur in a rather even frequency (Groth and Roelfs, 1982). Most cultures have several dominant genes for avirulence, and thus, the heterozygous F_1 cultures often have a narrow host range. However, selfing of heterozygous cultures occassionally results in cultures with a wide host range.

IV. Physiological Specialization

The use of race-specific resistance (see Chapter 13, this volume) to control wheat stem rust requires continued monitoring of the variation in the pathogen population for virulence (Volume I, Chapter 5). This has been and continues to be done annually in many countries. Published information appears regularly in Australia, Brazil, Canada, Egypt, India, Italy, Pakistan, and the United States. Currently, four systems for physiologic race identification in *P. graminis* f. sp. *tritici* are in use.

A. INTERNATIONAL SYSTEM

This system was developed by Stakman and Piemeisel and the most recent key was published by Stakman *et al.* (1962). The set uses 12 differential host cultivars (Table III) representing the three ploidy levels in wheat. These 12 differentials were used worldwide until about 1950, when sources of resistance not represented in them became widely used in commercial wheat cultivars. However, the international differential hosts are still used in many countries and still serve as a basis for communication. Physiologic races are based on combinations of host reaction classes, i.e., the resistant class is indicated by infection types 0, 0;, 1, and 2, mesothetic by infection type X, and susceptible by infection types 3 and 4. To 1983, 344 races have been described worldwide according to this system.

B. MODIFIED POTATO—*PHYTOPHTHORA INFESTANS* SYSTEM

This system was introduced in Australia and New Zealand and uses the international differential set (Table III) and then an ordered set of

Table III

International Differential Cultivars for Wheat Stem Rust and Their Genes for Resistance to *Puccinia graminis*[a]

Cultivar	*Sr* gene(s) known
Little Club	*LC*
Marquis	*7b, 18, 19, 20, X*
Reliance	*5, 16, 18, 20*
Kota	*7b, 18, 28, Kt'2'*
Arnautka	*9d*, plus two genes conditioning infection type X
Mindum	*9d*, plus a gene conditioning infection type X
Spelmar	*9d*, plus a gene conditioning infection type X
Kubanka	*9g*, plus a gene conditioning infection type X
Acme	*9g*, plus a gene conditioning infection type X
Einkorn	*21*
Vernal	*9e*
Khapli	*7a, 13, 14*

[a]Based on Luig *et al.* (1973) and Roelfs and McVey (1979).

Table IV

Additional Wheat Lines Used with the International Series as Differential Hosts for *Puccinia graminis* in Australia and New Zealand

Differential number	Host lines	*Sr* gene(s)
1	McMurachy or Eureka	*6*
2	Yalta	*11*
3	Gamenya	*9b*
4	Mengavi	*36*
5	Gala or Renown	*17*
6	Mentana	*8*
7	Norka	*15*
8	Festiguay or Webster	*30*
9	*Agropyron intermedium* derivative	*Agi*
10	Entrelargo de Montijo	Combination[a]
11	Barleta Benvenuto	8b[b]
12	Coorong Triticale	*27*

[a]Has a gene resulting in a low infection type (;) to pre-1954 strains and a gene resulting in a low infection type (2).

[b]Has a gene resulting in a low infection type (X).

differentials with sequential numbers (Table IV) that is increased as new sources of resistance are found to be effective in the separation of strains. The additional differential hosts were often lines with "single" effective genes for resistance, although the last two cultivars added are known to have at least two effective resistance genes. Nomenclature consists of the international race number (Stakman *et al.*, 1962), followed by ANZ for Australia–New Zealand and then the numbers of the additional differential hosts (Table IV) that are susceptible (Watson and Luig, 1963). Thus, 17 ANZ-2 is a strain of international race 17 that is virulent on *Sr11* and occurs in the Australia–New Zealand geographical area. Watson and Luig (1962) clearly point out that 17 ANZ-2 is *not* a subrace of race 17, as this artificially ranks some host genes as superior to others.

C. FORMULA METHOD

This system was developed and is used in Canada. The formula system is based mainly on differential hosts with a "single" gene for resistance to *P. graminis* (Table V) (Green, 1965). The avirulence/virulence formula is written for each culture, with the effective host genes listed first followed by a slash and then the ineffective host genes. A consecutive C number is assigned each different virulence formula: for example, C38(15B-1L) designates the virulence formula 6, 9a, 9b, 13, 15, 17/5, 7a, 7b, 8, 9d, 9e, 10, 11, 14, 35. The formula number (C38) indicates the 38th virulence formulas assigned a number in Canada. The old race designation was 15B-1L, which included the international race number and B-1L, which was a part of an old supplemental differential nomenclature that is no longer used but is retained to ensure continuity for those combinations for which it existed (Green, 1981).

D. CODED SETS

In the United States, three sets of four host lines, each with a "single" host gene for resistance, are used for physiological race identification (Roelfs *et al.*, 1982) (Table VI). The first set has the resistances present in the international differential set that were important in differentiating races of the asexual population in the United States.

Table V

Lines of Wheat Used as Differential Hosts for *Puccinia graminis* in Canada[a]

Host[b]	*Sr* gene
Prelude*6/Reliance	*5*
Mida-McMurachy-Exchange/6*Prelude	*6*
Na101/6*Marquis	*7a*
Chinese Spring/Hope	*7b*
Chinese Spring/Red Egyptian	*8*
Chinese Spring/Red Egyptian	*9a*
Prelude*4/2/Marquis*6/Kenya 117A	*9b*
H-44-24/6*Marquis	*9d*
Vernstein	*9e*
Marquis*4/Egypt NA95/2/2*W2691	*10*
Chinese Spring/Timstein	*11*
Prelude*4/2/Marquis*6/Khapstein	*13*
W2691*2/Khapstein	*14*
Prelude*2/Norka	*15*
Prelude/8*Marquis*2/2/Esp 518/9	*17*
Marquis*6/2/3*Steward/R.L. 5244	*22*
Agent	*24*
Eagle	*26*
WRT 238-5	*27*
Prelude/8*Marquis/2/Etiole de Choisy	*29*
Webster	*30*
Prelude*4/NHL II.64.62.1	*36*
W3563	*37*

[a]After Green (1981).

[b]The international differentials Marquis, Mindum, and Einkorn and the cultivars Manitou (*Sr5, 6, 7a, 9g, 12, 16,* plus), Selkirk (*Sr6, 7b, 9d, 17, 23, 2*), Sinton (*Sr* genotype unknown), and Neepawa (*Sr5, 7a, 9g, 12, 16,* plus) are tested.

The other two sets have resistance that have become important in more recent years. Races are designated by the international race number followed by a code indicating the virulence/avirulence formula for the culture. Thus, C-33(15B-1L) is similar to 15-TNM. The code for each set is based on a predetermined binary key of the 16 virulence/avirulence formulas possible within the set (listed from the formula of the possible pathogen phenotype avirulent on the four host lines to the last combination that is virulent on all four lines). The combinations of possible avirulent/virulent phenotypes are designated by letters B through T, omitting vowels (Roelfs and McVey, 1972).

Table VI

Lines of Wheat Used as Differential Hosts for *Puccinia graminis* in the United States[a]

Host lines	*Sr* gene
Set I	
ISr5-Ra	*5*
ISr9d-Ra	*9d*
Vernstein	*9e*
ISr7b-Ra	*7b*
Set II	
ISr11-Ra	*11*
ISr6-Ra	*6*
ISr8-Ra	*8*
ISr9a-Ra	*9a*
Set III	
W2691SrTt-1	*36*
W2691Sr9b	*9b*
W2691Sr13	*13*
W2691Sr10	*10*
Testers	
W2691Sr15NK	*15*
ISr16-Ra	*16*
Combination VII	*17* & *13*
Triumph 64	*Tmp*

[a]Bulks of cultures are tested on "universal" resistances *Sr22, 24, 25, 26, 27, 29, 31, 32, 33, 37, Gt,* and *Wld-1,* as well as *Sr30,* which is resistant to most cultures.

E. PHYSIOLOGICAL SPECIALIZATION: RYE STEM RUST

In initial studies by Levine and Stakman (1923), rye cultivars Rosen, Swedish, and Prolific were used. The cultivars Colorless and Dakold were added as differentials by Cotter and Levine (1932). Unfortunately, seed of these cultivars was apparently not maintained. Because rye is cross-pollinated and normally self-sterile, studies of physiological specialization are more difficult than in the *Triticum–P. graminis* system. Thus, a cultivar was considered resistant when 75% or more of the plants responded with infection types 0, 1, or 2. The response was rated mesothetic when 25–75% of infected plants had infection types 3 or 4, or when most of the plants have an infection type X, and susceptible

when more than 75% of the plants had infection types 3 or 4 (Cotter and Levine, 1932). A set of self-pollinated rye lines was developed that permits a more detailed investigation of virulence in *P. graminis* f. sp. *secalis* (Tan *et al.*, 1976). These ryes with "single" genes for resistance have been used in North America (Steffenson *et al.*, 1983) and Australia (Tan *et al.*, 1975) to study variation in *P. graminis* f. sp. *secalis*. The Australian work also included hybrids between *P. graminis* f. sp. *secalis* and *tritici* (Tan *et al.*, 1975), as well as *P. graminis* f. sp. *tritici* itself (Luig and Tan, 1978).

Another approach may be to move the rye resistance to a wheat line that is susceptible to *P. graminis* f. sp. *secalis* (Luig and Watson, 1976). Line E, W3498, derived from a cross of Gabo*3/Charter//Little Club. (Luig, 1983), is susceptible to *P. graminis* f. sp. *tritici* and most cultures of *P. graminis* f. sp. *secalis*.

V. Control

A number of methods are available to control wheat and rye stem rust, but none has been totally satisfactory alone. The earliest attempts involved religious practices. These practices varied between areas and existed as early as 1000 B.C. and continued into the first century A.D. (Chester, 1946). In the early 1600s, Worlidge recommended pulling a rope over the grain to knock off the dew. This practice continued until the 1900s in some areas (Chester, 1946). In France in the mid 1600s, laws requiring barberry eradication were passed. In the late 1800s, Farrer in Australia developed early-maturing wheat cultivars to escape the damage of rust (McIntosh, 1976). Resistant cultivars were known as early as 1841 (Chester *et al.*, 1951); however, breeding for resistance did not become common until the early 1900s. The two early successes were the transfers of stem rust resistance from Iumillo durum wheat to bread wheats by Hayes *et al.* (1936), and the transfer of resistance from emmer to bread wheat by McFadden (1930). The former resulted in the cultivar Thatcher, which is still a basis for much of the hard red spring wheats, and the latter resulted in Hope and H-44, which are among the most widely used sources of resistance (*Sr2*). Fungicides have been widely investigated for use in control of stem rust (Rowell, 1968), but only a few examples of commercial control have been documented (Mackie, 1935). *Darluca filum*, a hyperparasite, has been examined as a mechanism for rust control, and Chester *et al.* (1951) summarized this work. The integrated control program for wheat stem rust was re-

viewed for the United States by Rowell (1973) and for Canada by Green and Campbell (1979).

One of the most spectacular success stories in plant pathology and plant breeding is the complete control of wheat stem rust in the North American Great Plains for over 25 years. This highly destructive pathogen of wheat, which had periodically destroyed millions of hectares of wheat in a single year since the late 1880s, has been successfully overcome. The resistance of the cultivars released since the mid-1950s has endured, even though there has been a continual replacement by improved cultivars. Stem-rust-resistant cultivars have come from many different breeding programs; however, most have historically come from Agriculture Canada at Winnipeg; the United States Department of Agriculture in cooperation with the North Dakota State University at Fargo, with the University of Minnesota at St. Paul, and with the University of Nebraska at Lincoln; CIMMYT (Centro Internacional Mejoramiento de Maíz y Trigo) at Mexico City; and the University of Sydney in New South Wales. These resistant cultivars are generally composed of multigenic resistances that were selected in the field by using a very heavy inoculum density of pathogen cultures differing widely in their phenotypes for virulence. These resistant cultivars have reduced the pathogen population, so that in the past 5 years few samples of stem rust have been found in commercial wheat fields in North America. Nearly all the collections for the annual race surveys in Canada, the United States, and Mexico are made from trap plots, susceptible cultivars in nurseries, or from non-*Triticum* hosts. This reduction in inoculum in turn makes the host resistances used more effective and also reduces the total numbers of pathogen mutations that are likely to occur.

A. CULTURAL METHODS

Currently, cultural methods largely depend on the use of early maturing cultivars and early planting of spring wheats. However, early planting of fall-sown grains may actually increase the chance for fall infection and overwintering of the rust in milder climates. The environmental conditions that favor wheat and rust development are similar. Avoiding excess nitrogen applications and frequent light applications of irrigation water are generally helpful in controlling stem rust. In areas where the disease oversummers, destruction of volunteer wheats and other susceptible grasses several weeks before planting also reduces inoculum level and delays initial infection. Where both winter

and spring wheat are grown in the same area, separating these crops by space or by another type of crop can delay the spread between fields. Diversity in the cultivars grown on a farm and spacing between fields of wheat can provide substantial benefits (see Chapter 13, this volume).

B. BARBERRY ERADICATION

Eradication of the alternate host was started in Rouen, France, in 1660, over 100 years before science showed a relation between stem rust and barberry. Reviews of the successful programs in Denmark and the United States were reported on by Hermansen (1968) and Roelfs (1982), respectively. These studies revealed four effects of barberry eradication on stem rust epidemics: (1) delayed disease onset, (2) reduction in initial inoculum level, (3) decreased number of pathogen races, and (4) stabilization of pathogen phenotypes. The available evidence indicates that much of this change may result from eliminating the sexual population of *P. graminis* and a corresponding increase in the importance of the asexual population.

1. Delayed Disease Onset

Aeciospores are produced locally in large numbers early in the spring. In contrast, exogenous inoculum arrives only after urediospores are produced elsewhere. For effective inputs of exogenous inoculum, weather conditions must be favorable for urediospore production, liberation, and long-distance transport, and the arriving urediospore must be deposited on a susceptible host genotype when conditions are favorable for infection. The difference in date of disease onset before and after barberry eradication on a regional basis is now difficult to document in many cases, as data on date of disease onset were not collected from infection centers near individual barberry bushes with regularity. In Minnesota, Stakman and Harrar (1957) reported the normal severity for stem rust of wheat in commercial fields was trace to 8% on July 8. From 1965 through 1982, traveling the same route, Roelfs (1982) found only traces of rust, and in most recent years no rust was found even on trap plots of susceptible cultivars.

2. Reduction in Initial Inoculum Level

A rust-infected barberry bush can produce around 60×10^9 spores daily, thus resulting in heavy infection in the vicinity of the infected

barberry bush (see Section II,C). An exogenous inoculum source is unlikely to provide an equal amount of inoculum to areas remote from it. Exogenous inoculum usually is not concentrated at a point but is generally dispersed in transport and then deposited over a large area (Rowell and Roelfs, 1971). Thus, for effective dispersal of large amounts of exogenous inoculum, environmental conditions must be favorable for disease increase in the source area, for spore transport, for spore deposition, and for infection in the target area. The spores must then possess virulence for the cultivars in the target area. Rarely are these conditions all met, and the number of epidemics are few. However, the epidemics of 1935, 1937, 1953, and 1954 in the United States and Canada show the potential threat of exogenous inoculum (Stakman and Harrar, 1957).

3. *Decreased Number of Pathogen Phenotypes*

Sexual recombination as the major source of new combinations of virulence genes is eliminated with eradication of the barberry. As virulence is normally recessive, mutations for virulence are not always expressed. Parasexualism seems to result in few recombinations that exist in the natural population (Luig, 1977). In one sexually reproducing population, 1 phenotype recombinant was found per 4.2 isolates but only 1 per 148 isolates in an asexually reproducing population (Roelfs and Groth, 1980).

4. *Stabilization of Pathogen Phenotypes*

Not only has elimination of the sexual cycle reduced the number of pathogen phenotypes for virulence, it also has stabilized them over time. This can be demonstrated by comparing the years 1918 through 1921, when a different race (pathogen phenotype) predominated in the race survey each year, with the 48 years since 1934, when only four races dominated the annual populations and two races predominated in 46 of the years (Roelfs, 1982). These four races represent more than four pathogen phenotypes, but a single phenotype has predominated for periods greater than 10 years. Early in the barberry eradication program, great differences existed among the races found between years; thus, in 1918, 28 races were identified; six of these races were not found in 1919, but three different races were found (Roelfs, 1982).

Removal of barberry has resulted in reduced yield losses and made it possible to successfully breed for resistance to stem rust. Many of the resistances currently used could not withstand the very high inoculum densities near an infected barberry bush (for example *Sr36*, *Sr2*, Thatcher, and so forth).

C. RESISTANCE IN THE CROPS

Certain combinations of single host genes for resistance have effectively controlled stem rust for many years. Many examples could be given; however, the story starts with the introduction of the cultivar Selkirk (*Sr6, 7b, 9d, 17, 23, 2*) which terminated the 15B epidemics of the early 1950s, in the northern Great Plains of North America. Selkirk was widely grown until the mid 1960s, when it was replaced by higher yielding, leaf rust-resistant cultivars. Selkirk has remained resistant in field plots through 1983. The cultivars Era (*Sr5, 6, 12, 17,* plus) and Waldron (*5, 11, Wld-1*) were introduced over 10 years ago; each has been grown on over 1 million hectares annually for 10 years, and they have remained as resistant to stem rust as they were when released. The current list of designated host genes for resistance, their sources, low-infection types in seedlings, and comments on their response or effectiveness under field conditions are given in Table VII. Resistance of the race-specific type is discussed in detail in Chapter 15, this volume.

Stomatal exclusion and size of collechyma bundles were proposed as types of morphological resistance in wheat to *P. graminis* (Hart, 1931). They now are both thought to be unlikely mechanisms of resistance. *Puccinia graminis* normally penetrates closed stomata (Volume I, Chapter 10). Webster, the original cultivar studied, has small collenchyma bundles, but cultures virulent on it are known. The small uredia on Webster with most avirulent cultures is now known to be due to *Sr30* (Knott and McIntosh, 1978).

The resistance not yet shown to be race-specific (Chapter 16, this volume) has attracted attention in recent years. Whether this resistance is a unique response or is just a result of combinations of factors that are too small to measure with current technology is unknown in most cases.

Whether a resistance is of the specific or nonspecific type, it would appear that it restricts the disease in one of four ways. Several of the single genes listed in Table VII function in at least two ways.

1. Reduction in Number of Successful Infections

Host plants often differ in receptivity to the fungus; equal inoculum densities result in different numbers of uredia. This receptivity can be measured in the field by inoculating plants with a single heavy inoculum density and taking notes 14 days later (Rowell and McVey, 1979). The reduction may be total as with cultures avirulent to *Sr5* on hosts

Table VII

Known Host Genes for Resistance and Their Response to *Puccinia graminis* f. sp. *tritici*[a]

Sr gene	Source[b]	Response to an avirulent culture: Seedling infection type[c]	Response to an avirulent culture: Adult plant response[d]
2	*T. durum*	None	Few large uredia at nodes, and in spike
5	*T. aestivum*	0	Immune
6	*T. aestivum*	;	Highly resistant
7a	*T. aestivum*	23C	Moderately susceptible
7b	*T. aestivum*	2	Moderately susceptible
8	*T. aestivum*	2	Moderately susceptible
9a	*T. aestivum*	2⁻, 23	Moderately susceptible
9b	*T. aestivum*	2⁺	Moderately susceptible
9d	*T. durum*	;1	Moderately resistant
9e	*T. dicoccum*	;1	Moderately resistant
9f	*T. aestivum*	;2⁻	Rare, culture not evaluated
9g	*T. durum*	2⁻	Moderately resistant
10	*T. aestivum*	;1⁺N	Moderately susceptible
11	*T. aestivum*	;2⁻,2⁺3⁻	Moderately resistant
12	*T. durum*	0;X⁻	Moderately resistant
13	*T. durum*	2⁺	Moderately susceptible
14	*T. durum*	12C	Moderately susceptible
15	*T. aestivum*	;1⁺N	Nearly susceptible
16	*T. aestivum*	2	Moderately susceptible
17	*T. durum*	;1N	Moderately resistant
18	*T. aestivum*	;2⁼	Rare, culture not evaluated
19	*T. aestivum*	1⁻	Rare, culture not evaluated
20	*T. aestivum*	2⁼	Rare, culture not evaluated
21	*T. monococcum*	;1⁼	Resistant
22	*T. boeoticum*	0;	Moderately susceptible
23	*T. aestivum*	23C	Nearly susceptible
24	*A. elongatum*	2⁻	Moderately susceptible
25	*A. elongatum*	2⁻	Moderately susceptible to susceptible
26	*A. elongatum*	;2⁻	Resistant
27	*S. cereale*	0;	Highly resistant
28	*T. aestivum*	;	Rare, culture not evaluated
29	*T. aestivum*	2⁺2⁻	Moderately susceptible
30	*T. aestivum*	2⁻2	Moderately susceptible
31	*S. cereale*	0;	Resistant
32	*Ae. squarrosa*	2⁻	Moderately susceptible
33	*Ae. squarrosa*	2	Moderately susceptible
34	*T. aestivum*	3C	Moderately susceptible

(*continued*)

Table VII (*Continued*)

Sr gene	Source[b]	Response to an avirulent culture: Seedling infection type[c]	Response to an avirulent culture: Adult plant response[d]
35	*T. monococcum*	0;	Resistant
36	*T. timopheevii*	0,0;1+,40;	Immune, fewer uredia
37	*T. timopheevii*	0;	Highly resistant
LC	*T. aestivum*	2−	Rare, culture not evaluated
Gt	*T. aestivum*	2	Moderately resistant
dp-2	*T. durum*	2	Resistant
X	*T. aestivum*	23C	Moderately susceptible
McN	*T. aestivum*	2−;	Rare, culture not evaluated
Kt'2'	*T. aestivum*	2	Rare, culture not evaluated
Wld-1	*T. aestivum*	2=C	Resistant to moderately resistant
Tt-3	*T. timopheevii*	0,;1+C	Resistant
U	*T. aestivum*[e]	21CN	Moderately susceptible
H	*T. durum*[f]	2C	Moderately susceptible

[a]Updated from Roelfs and McVey (1979).

[b]*Triticum* = *T.*, *Agropyron* = *A.*, *Aegilops* = *Ae.*, *Secalis* = *S.*

[c]Infection types at 18°C (plants with *Sr6*, *10*, *12*, *15*, and *17* are more susceptible at higher temperatures, whereas plants with *Sr13* are more resistant); variation is encountered with host genetic background and ploidy level also (Luig and Rajaram, 1972).

[d]Many host resistances are less effective at high temperatures, high inoculum densities, and at plant maturity. Variations also occur with different host genetic backgrounds.

[e]Gene from Red Egyptian other than *Sr6*, *8*, and *9a* (Loegering, 1968).

[f]Gene from H-44, other than *Sr7b*, *9d*, and *17*. *SrH* was the cause of the differences in Canadian and United States race survey data in the 1970s (Green and Dyck, 1979).

possessing *Sr5*, or partial as with *Sr36* (*Tt-1*) (Rowell, 1981) and *Sr2* (Sunderwirth and Roelfs, 1980). Reduction can also be the result of several host factors, as in Thatcher wheat (Nazareno and Roelfs, 1981). Even among susceptible host cultivars, receptivity varies widely under field conditions (Rowell and McVey, 1979).

2. *Lengthened Latent Period*

Although often considered to be due to nonspecific resistance, a lengthened latent period may result from race-specific resistance. The *Sr36* gene derived from *T. timopheevii* conditions an increased latent period for penetrants that result in successful infections (Rowell, 1981). In the wheat–*Puccinia graminis* system, there are fewer known genotypes with long latent periods than in some other host–rust systems. However, there is a trend for longer latent periods with infec-

tions on many host genotypes as the plant ages (Sunderwirth and Roelfs, 1980); this effect often declines or even reverses itself with the start of host senescence.

3. Reduction in Size of Sporulating Area

Most resistance of the race-specific type listed in Table VII functions by reducing the sporulating area per lesion. Some resistances like *Sr9e* reduce the sporulating area until few spores are produced per lesion, whereas others like *Sr5* result in no sporulation and some like *Sr29* provide little reduction in numbers of spores produced. The amount of reduction in spore production due to a gene(s) for resistance is often affected by temperature, light, ploidy level of host, and host growth stage.

4. Reduction in Duration of Uredial Sporulation

Although some resistance genes seem to reduce the duration of uredial sporulation in infections of seedling leaves, I know of no data from adult plants that would indicate that uredia produce urediospores over varying time periods. However, it may be assumed such resistance exists but remains unreported owing to interactions with host age and environment.

D. FUNGICIDES

The use of fungicides for control of stem rust has been studied for many years (Rowell, 1968). However, chemical control has played a very minor role in stem rust control. The reasons are at least threefold: (1) the effectiveness of the host resistance, (2) the very high rate of disease increase for wheat stem rust under ideal conditions, and (3) the relatively low economic return per hectare of wheat in comparison with the cost of fungicide applications.

E. BIOLOGICAL CONTROL

The hyperparasite of rust, *Darluca filum* (Biv.) Cast., has been widely considered (Chester *et al.*, 1951); however, it seems currently to offer little promise as the rust must be present to have a buildup of the hyperparasite. Another hyperparasite, *Aphanocladium album*, is now being evaluated on a field scale, but its potential is currently unknown. Although *Verticillium niveostratosum*, *V. fungicola*, and *Cephalos-*

porium acremonium were found to be greenhouse parasites of rust, they were thought to have little potential as a practical means of controlling stem rust (Chester, 1946).

VI. Losses

Stem rust develops at warmer temperatures (30°C optimum) than the other rust diseases of wheat and rye. Thus, it is most frequently a disease of the reproductive portion of the host life cycle. Occasionally stem rust can become severe on early sown, fall-planted wheat or on irrigated wheat in tropical areas. Infection by stem rust of seedling wheat or rye under favorable environmental conditions can result in death of tillers or entire plants. A tiller of an adult wheat plant has a surface area of approximately 150 cm^2 including leaf and stem tissue. A disease severity of 100% (6.7 infections/cm^2) destroys the tiller (Rowell and Roelfs, 1976). Severe amounts of disease can halt plant growth or even kill the plant by reducing the photosynthetic area, causing a loss of nutrients and water and disrupting the plant transport system (see Volume I, Chapter 16). Restricted growth often results in small shriveled grain, weakened stems that break or lodge, and in severe cases the death of the plant.

A. Reduction in Photosynthetic Area

Lesions of rust can occupy a significant portion of the host plant tissue. The tissues affected are usually the flag leaf, peduncle, glumes, and awns, the very parts that are the source of most of the nutrients that are transported to the developing grain.

B. Loss of Nutrients and Water

The rupture of the plant epidermal cells by the rust fungus results in a loss of water from the plant. Because the pathogen also uses both water and nutrients from the plant to produce the large volume of urediospores daily, the plant suffers added stress. Infection at an early growth stage results in decreased availability and production of nutrients for plant growth. An infected plant also has less root growth, which aggravates the imbalance in normal water requirements. Such plants are more susceptible to winterkill, produce fewer tillers, have smaller heads, and occasionally have decreased spikelet fertility.

C. DISRUPTION OF NUTRIENT TRANSPORT

Stem rust is characterized by development of uredia on leaf sheaths and peduncle tissue. The fungus often penetrates through the tissue of the true stem. The rupture of the plant tissue by fungus can disrupt transport of nutrients to the roots and cause premature death of the roots (Bushnell and Rowell, 1968). Shriveled kernels result from disruption of nutrient transport to the filling grains (Calpouzos *et al.*, 1976).

D. STEM BREAKAGE AND LODGING

When disease is extremely severe on a portion of the stem, the straw may break, causing the plant spike to break over or fall to the ground. With mechanical harvesting, broken and lodged plants often have the spike below the cutter bar level, making the grain impossible to harvest economically.

E. ESTIMATION OF LOSSES

The several models developed to estimate losses due to stem rust were reviewed by Calpouzos *et al.* (1976). A table relating rust severity at different crop stages to loss was developed by Kirby and Archer (1927). Greaney (1935) found the average loss in spring wheat due to stem rust was 5.4% (range 3.1–9.7) for each 10% of terminal rust severity. Kingsolver *et al.* (1959) related loss in yield to the growth stage at which a 1% disease severity (10 pustules/tiller) occurred. The area under the disease progress curve (y = disease severity and x = days from heading) was used by Buchenau (1970) to predict losses due to stem rust. Calpouzos *et al.* (1976) related loss due to stem rust to the host stage when disease started and the rate at which the disease increased. These models all need more evaluation by using current agricultural practices and cultivars. The model by Calpouzos *et al.* (1976), although the most complex model, varied from extremely accurate to very inaccurate in later tests (Rowell, 1982).

F. WORLDWIDE LOSSES

Losses in Europe due to wheat stem rust were summarized by Hogg *et al.* (1969). They recorded only two mild epidemics in the 1960s, one in Czechoslovakia in 1962 and one in Portugal in 1960. At least seven

nation-wide epidemics occurred in the 1920s. In the United States, no major national epidemics have occurred since 1954 (Roelfs, 1978).

Although stem rust is an important disease in Asia, Australia, Africa, and South America, summaries of losses from these areas are unavailable from published sources except for a summary of epidemics by Chester *et al.* (1951).

VII. The Future

Wheat stem rust is the most researched host–pathogen system in agriculture. However, much remains to be learned, in the areas of both applied and basic research. Probably the most elusive property has been the physiologic basis of resistance. Many leads have been followed but without success (Volume I, Chapter 7). The effect of many individual host genes has been studied on seedlings, but their effects on epidemics have not been carefully documented. The effects of combinations of these genes are largely unknown. Most cultivars have some level of resistance in comparison with the most susceptible cultivars known, that is, Line E or Morocco. The genetic basis of this resistance, however, is still unknown.

The mechanism by which the pycniospore fuses with the receptive hyphae, the migration of the nuclei following the fusion, the penetration of the barberry leaf by the infection peg of the basidiospore, and the germination of the teliospore all need clarification.

Genetic studies of the pathogen remain in their infancy. The only factors studied to date are spore color and virulence. Mutation rates for important virulence genes are generally unknown. Although other differences exist among pathogen cultures, they are generally ignored. Certainly future studies will need to consider aggressiveness and adaptation of biotypes. The use of adult plant resistance may necessitate evaluation of cultures on other than seedlings.

Studies of pathogen development must be done for large regions where different crops are grown and cultivars with different resistances are grown, that is, the real world of agriculture. As models of the pathogen–host–environment system are built, values for individual host resistance and pathogen virulence genes will have to be altered as the host growth stage and temperature change. Certain host genes in combination as well as certain pathogen genes in combination will not be equal to the best component (epistasis) or the sum of the components (additive) as is currently generally assumed.

Will stem rust again result in serious epidemics? Yes, the pathogen will change through mutation and selection. Yes, favorable environmental conditions will occur. However, genes for resistance exist and can be bred into well-adapted, high-yielding, and high-quality cultivars. Yet when the environment is ideal and inoculum density high, even resistant cultivars can fail (Roelfs *et al.*, 1972). It appears that a continued development of cultivars with a combination of different types of resistance can reduce the inoculum and prevent the pathogen population from increasing and thus can avoid catastrophic epidemics in the near future.

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